CLINICAL REVIEW

Volume 6 Issue 1

Modern Ventilator Management in Surgical/

Trauma Patients

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ABSTRACT

Physicians have a lot to consider when they place a patient on the ventilator. This article reviews the literature and provides the physician with a clear guide to managing the ventilator in the ICU. This manuscript is meant to be a practical guide to managing the ventilator at the patient's bedside.

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KEYWORDS

Vent, Ventilator, Surgery, Ventilator Management, Respiratory Failure, PEEP, SIMV, APRV, pneumonia, sepsis, assist control, ARDS, CPAP, ventilator induced lung injury, volutrauma, tidal volume, weaning, weaning from ventilator

SUMMARY

Ventilator management is a staple of critical care medicine. The consequences of poor ventilator management are significant complications, including pneumonia, sepsis, and death.¹ Therefore, it is crucial that physicians understand how to use mechanical ventilation to maximally benefit their patients. This review is intended to be used as a guide to ventilator management for residents, physicians, and other health-care professionals who do not typically care for ventilated patients on a daily basis.

WHO NEEDS MECHANICAL VENTILATION?

Normally, ventilation and perfusion are perfectly matched. Once this balance becomes tilted towards perfusion or ventilation, the patient can develop respiratory distress or failure. There is no one specific test or study that definitely indicates a requirement for intubation and mechanical ventilation. Instead, a constellation of symptoms and signs must be considered. Patients who require high levels of airway pressure in the trachea for oxygenation or ventilation should be intubated. Patients who are struggling to breathe may have high airway pressure due to an extrinsic pathology, such as a pneumothorax, or an intrinsic problem, such as pneumonia. In addition, patients who are unable to protect their airway should be intubated, and patients who, for whatever reason, cannot clear their secretions should also be intubated. Finally, patients who have developed an upper airway obstruction that may be bypassed by an endotracheal tube or a tracheostomy tube should be intubated and placed on mechanical ventilation. In sum, any patient who cannot adequately oxygenate or ventilate should be intubated. This review will not delve into the gray area of mechanical ventilation versus non-invasive positive pressure ventilation.

It would be ideal if the lungs had perfectly matched perfusion and ventilation in every alveolus, but unfortunately, this is not the case. The position of a patient affects both ventilation and perfusion. For example, if a patient is standing, the apex of the lung has a high level of ventilation with a low level of perfusion.² The term "dead space" refers to a lung that is ventilated with little or no perfusion. Dead space can occur anywhere in the lung parenchyma where there is ventilation with no perfusion. No significant gas exchange can take place in this area



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of the lung. Because of gravity, perfusion is highest in the bases of the lungs in an upright individual. Perfusion and ventilation are relatively ideal in the mid-portions of the lungs, as these regions have optimal gas exchange. In the supine position, the posterior lungs have the highest level of perfusion and low levels of ventilation. Once again, the midportions or centers of the lungs have the ideal match between ventilation and perfusion.² In patients with adult respiratory distress syndrome, CT scans of the chest reveal consolidation in the posterior aspect of the lung. Improved oxygenation can be achieved by simply pronating the patient.

VENTILATOR MODES

A series of recent studies have narrowed the choices between a dizzying array of ventilator modes. Synchronized intermittent mandatory ventilation was once thought to be the ideal ventilatory mode. Basically, in this mode, the physician sets the patient's tidal volume, rate (breaths per minute), percentage of oxygen, positive end-expiratory pressure (PEEP), and pressure support. The patient can breathe at whatever rate he/she wants, and the patient will receive the set number of machine breaths plus pressure support breaths. For example, if a patient ideally breathes at 22 times per minute and the ventilator is set for 10 breaths per minute, the patient will receive 10 machine breaths plus 12 pressure-supported breaths. The tidal volume for these pressure support breaths can be increased or decreased depending on the amount of pressure added to each breath. In theory, careful monitoring of the patient and adjustment of the pressure support as well as the respiratory rate allows the patient to be smoothly weaned from the ventilator. This mode has fallen out of favor, as it was shown to have no added benefits over assist-control ventilation (ACV).^{3,4}

ACV is a simple, straightforward mode of ventilation in which the rate, tidal volume, PEEP, and fraction of inspired oxygen (FiO2) are set by the health-care provider. A patient can breathe at a rate greater than the rate set on the ventilator, but with each breath, the patient receives the tidal volume set by the physician. For this mode of ventilation, either volume or pressure control is used. Most physicians use volume control because without careful monitoring by respiratory therapists, nurses, and physicians, pressure control can lead to severe complications.

When intubated, lung compliance can change for any number of reasons, including secretions, atelectasis, and pneumonia. If pressure control is used, patients receive a larger or smaller tidal volume, depending upon lung compliance. If a particular pressure is selected and no adjustments are made for these issues, a patient may develop atelectasis. Atelectasis increases a patient's pulmonary compliance, leading to receipt of a lower tidal volume for a given pressure setting. Without intervention, the patient may develop progressive atelectasis and increased respiratory distress in spite of being on the ventilator. Ultimately, this can progress to extreme tachypnea and cardiovascular collapse without early intervention. For this reason, using a pressure mode of ACV is often discouraged. Thus, in ACV, volume control should be the main mode of mechanical ventilation used by the average physician in the vast majority of their patients.^{5,6}

SETTING THE VENTILATOR

Almost 20 years ago, a randomized multicenter study conducted as part of the National Institutes of Health-National Heart, Lung, and Blood Institute (NIH-NHLBI) Acute Respiratory Distress Syndrome (ARDS) Network investigated the difference between low tidal volumes (6 ml/kg) and traditionally high tidal volumes (12 ml/kg). This famous study was stopped early upon observation of increased mortality in patients with high tidal volumes.⁷ A subsequent retrospective study in which patients who were ventilated with low tidal volumes and low plateau pressures (0.5-second pause after inspiration) were found to have lower mortality rates than their historical controls, validating the findings of the ARDS Network study. Based on these studies, setting the ventilator at 6 ml per kg of ideal body weight is now standard of care.

Patients who were recently intubated should be placed on 100% oxygen until their respiratory status stabilizes; however, physicians must understand that oxygen is toxic to normal pulmonary tissue.^{9,10} One study found that within a couple of hours, more



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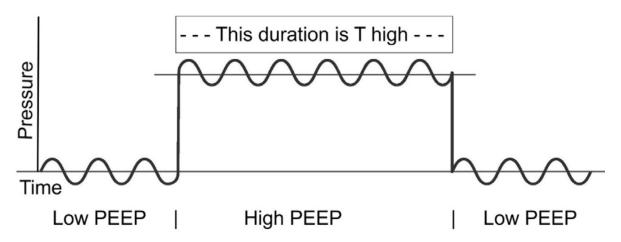


FIGURE 1. This schematic shows pressure vs. time in APRV. The black line indicates the patient's spontaneous breathing during both low and high PEEP.

than 60% of healthy volunteers developed chest pain after being placed on 95% oxygen.¹¹ In addition, an alveolar-capillary leak, which is associated with shortterm exposure to high-dose oxygen, was described in some of these individuals. Therefore, it is imperative for physicians to wean patients down to reasonable levels of oxygen quickly. While the lowest dose of oxygen that is well-tolerated by the human lungs is unknown, a reduction of the concentration of oxygen to less than 50% as quickly as possible is prudent.

PEEP

PEEP was originally developed in the mid-1930s¹² and was widely adopted in the 1970s and 1980s as a means for improving oxygenation and preventing atelectasis. There are, however, important pros and cons of using PEEP. Adding positive pressure at the end of expiration prevents the collapse of alveoli and ensures their continued participation in air exchange, leading to improved oxygenation.¹³ Therefore, in diseases like ARDS, PEEP has the ability to open up an atelectatic lung.¹⁴ PEEP can also improve gas exchange in cardiogenic pulmonary edema.¹⁵ Finally, PEEP can increase the functional residual capacity of lungs.^{16,17}

Unfortunately, PEEP can also lead to complications. Excessive PEEP can cause hypoxia and hypercapnia due to impaired capillary gas exchange and increased dead space ventilation, respectively.¹⁸ PEEP is also associated with detrimental effects on the cardiovascular system, primarily via a decrease in venous return to the heart.¹⁹ PEEP may cause increased intra-abdominal pressure, leading to decreased splanchnic circulation as well as decreased urine output.²⁰ In patients with severe head injuries, high PEEP is clearly associated with an increase in intracranial pressure and worse outcomes.²¹ High PEEP, as well as high tidal volumes and increased respiratory rates, are associated with volutrauma or ventilator-induced lung injury. Overdistention of the lungs is associated with worse outcomes. High airway pressures can lead to alveolar overdistention, which can lead to lung rupture and pneumothorax. More importantly, this overdistention can release pro-inflammatory mediators that may worsen gas exchange, leading to increased pulmonary edema, more time on the ventilator, and increased risk of mortality.22,23,24

Most health-care professionals agree that a PEEP of 5 cm of H2O is probably physiologic. In patients who are prone to atelectasis, specifically trauma patients with pulmonary injuries and morbidly obese patients, a PEEP of 8 cm of H2O is optimal to prevent atelectasis. If the patient is still hypoxic or on greater than 50% oxygen, then the PEEP can be slowly increased by 2 cm of H2O every 15-30 minutes with concomitant decreases of the FiO2. Most patients should be able to adequately oxygenate between a PEEP of 8 and 14 cm of H2O. During adjustment of these parameters by the physician, plateau pressures must be monitored and adjusted to less than 30 cm of H2O.



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ADVANCED RESPIRATORY FAILURE

Although there are multiple advanced modes of mechanical ventilation designed to improve oxygenation, airway pressure release ventilation (APRV) is currently popular and warrants discussion. To understand APRV, one should first understand continuous positive airway pressure (CPAP). With CPAP, a patient spontaneously breathes through a face mask, which has an outflow value (expiratory) that maintains PEEP. CPAP, therefore, increases the patient's functional residual capacity.²⁵

APRV is simply a combination of a low CPAP and a high CPAP (Figure 1)^{25,26} and has several theoretical advantages.

First, it is a pressure mode that delivers a steady pressure throughout inspiration and in theory, should hold open alveoli a little longer than a volume mode. Secondly, APRV allows for spontaneous breathing. Several studies suggest that spontaneous breathing allows for better ventilation along the diaphragm and therefore less atelectasis.^{27,28} Theoretically, this benefit makes APRV an ideal mode to combat ARDS, especially since APRV combines several mechanisms to improve both oxygenation and ventilation.

Setting the ventilator with APRV requires both a high pressure and a low pressure. The high pressure (P high) ranges from 20 to 30 cm of H2O. The goal of the P high is to completely inflate the lungs without overdistention. The low pressure (P low) is usually set between 0 and 5 cm of H2O. Time (T) high is the amount of time that the ventilator is at P high and is usually set at 4-6 seconds. T low is the amount of time that the ventilator is at P low and is normally set at 0.2-0.8 seconds. Tidal volume is the difference between P high and P low.²⁴ When placing a patient on this mode of ventilation, instantaneous changes in oxygenation or ventilation should not be expected, as this effect takes time. In fact, it may take between 12 and 24 hours before improvements in the patient's oxygenation and/or ventilation are observed.

In patients with severe respiratory failure/ARDS, APRV has theoretical advantages over other advanced modes of mechanical ventilation; however, APRV has not been shown to be consistently beneficial in treating all patients with severe respiratory failure/ARDS.²⁹ While this mode of ventilation is not a panacea, it can be helpful for some patients. Currently, it is not known which patients with severe respiratory failure/ARDS will benefit from APRV.

DISCONTINUATION OF THE VENTILATOR

Before weaning the patient from the ventilator, the patient should be hemodynamically stable. The physician must also determine whether the patient's secretions are manageable off the ventilator. When it is determined that a patient is ready to be weaned from the ventilator, several methods may be used. The patient can first be placed on spontaneous intermittent mandatory ventilation, and then the rate can be gradually decreased until the patient is breathing completely without assistance. The second method involves placing the patient on pressure support ventilation. By slowly decreasing the amount of pressure support, the patient begins to gradually breathe more independently. Once the patient is on a pressure support of 5 cm of H2O, the patient can be extubated. The third method requires a spontaneous breathing trial. The patient is placed on a T-piece and allowed to breathe spontaneously for 2 hours. If the patient tolerates the spontaneous breathing trial, the patient can then be extubated. If the patient does well on the spontaneous breathing trial but is not quite ready to be extubated, a spontaneous breathing trial may be repeated later in the day.

Esteban and colleagues investigated methods of weaning from the ventilator.³⁰ In a prospective, randomized clinical trial, a once-daily spontaneous breathing trial was found to be significantly better than any of the other weaning methods. Simply put, patients were weaned off the ventilator more quickly via a once-daily spontaneous breathing trial. Therefore, once-daily spontaneous breathing trials should be the weaning method of choice. Such a spontaneous breathing trial should consist of placing a patient on CPAP at 5 cm, a pressure support of 5-8 cm, and a FiO2 of 40% or less.

SUMMARY

Based on the literature, best practices for managing and weaning from ventilation have been established.



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After intubation, the patient should be placed on assist control with a tidal volume of 6 ml/kg. The FiO2 should start at 100% and be reduced as quickly as possible. The goal should be a FiO2 of less than 50%. PEEP should be increased in order to maintain oxygen saturation at 92% or greater. If the patient develops ARDS, advanced ventilator modes, such as APRV, may be necessary. APRV has shown promise in improving both oxygenation and ventilation of patients with severe respiratory failure/ARDS. Finally, when the patient is ready to be weaned from the ventilator, a once-daily spontaneous breathing trial is the superior method for liberating the patient from the ventilator.

AUTHOR AFFILIATIONS

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REFERENCES

- Esteban A, Anzueto A, Frutos F, Alía I, Brochard L, Stewart TE, Benito S, Epstein SK, Apezteguía C, Nightingale P, Arroliga AC. Characteristics and outcomes in adult patients receiving mechanical ventilation: a 28-day international study. JAMA. 2002 Jan 16;287(3):345-55.
- West JB, Dollery CT. Distribution of blood flow and ventilation-perfusion ratio in the lung, measured with radioactive CO2. J App Physiol. 1960 May 1;15(3):405-10.
- 3. Groeger JS, Levinson MR, Carlon GC. Assist control versus synchronized intermittent mandatory ventilation during acute respiratory failure. Crit Care Med. 1989 Jul;17(7):607-12.
- 4. Tobin MJ. Mechanical ventilation. N Engl J Med. 1994 Apr 14;330(15):1056-61.
- 5. Tobin MJ. Principles and practice of mechanical ventilation. McGraw Hill Professional; 2013.
- Bendixen HH, Whyte H, Laver MB. Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation: a concept of atelectasis. N Engl J Med. 1963 Nov 7;269(19):991-6.
- 7. Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N

Engl J Med. 2000 May 4;342(18):1301-8.

- 8. Kallet RH, Jasmer RM, Pittet JF, Tang JF, Campbell AR, Dicker R, Hemphill C, Luce JM. Clinical implementation of the ARDS network protocol is associated with reduced hospital mortality compared with historical controls. Crit Care Med. 2005 May 1;33(5):925-9.
- 9. Klein J. Normobaric pulmonary oxygen toxicity. Anesth Analg. 1990 Feb 1;70(2):195-207.
- 10. Deneke SM, Fanburg BL. Normobaric oxygen toxicity of the lung. N Engl J Med. 1980 Jul 10;303(2):76-86.
- Davis WB, Rennard SI, Bitterman PB, Crystal RG. Pulmonary oxygen toxicity: early reversible changes in human alveolar structures induced by hyperoxia. N Engl J Med. 1983 Oct 13;309(15):878-83.
- Barach AL, Martin J, Eckman M. Positive pressure respiration and its application to the treatment of acute pulmonary edema. Ann Intern Med. 1938 Dec 1;12(6):754-95.
- 13. Gregory GA, Kitterman JA, Phibbs RH, Tooley WH, Hamilton WK. Treatment of the idiopathic respiratory-distress syndrome with continuous positive airway pressure. N Engl J Med. 1971 Jun 17;284(24):1333-40.
- Gattinoni L, Caironi P, Cressoni M, Chiumello D, Ranieri VM, Quintel M, Russo S, Patroniti N, Cornejo R, Bugedo G. Lung recruitment in patients with the acute respiratory distress syndrome. N Engl J Med. 2006 Apr 27;354(17):1775-86.
- 15. Matamis D, Lemaire F, Harf A, Teisseire B, Brun-Buisson C. Redistribution of pulmonary blood flow induced by positive end-expiratory pressure and dopamine infusion in acute respiratory failure. Am Rev Respir Dis. 1984 Jan;129(1):39-44.
- Rose DM, Downs JB, Heenan TJ. Temporal responses of functional residual capacity and oxygen tension to changes in positive end-expiratory pressure. Crit Care Med. 1981 Feb;9(2):79-82.
- Gattinoni L, Mascheroni D, Torresin A, Marcolin R, Fumagalli R, Vesconi S, Rossi GP, Rossi F, Baglioni S, Bassi F, Nastri G. Morphological response to positive end expiratory pressure in acute respiratory failure. Computerized tomography study. Intensive Care Med. 1986 May 1;12(3):137-42.
- 18. Hasan FM, Beller TA, Sobonya RE, Heller N, Brown



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GW. Effect of positive end-expiratory pressure and body position in unilateral lung injury. J App Physiol. 1982 Jan 1;52(1):147-54.

- 19. Marini JJ, Culver BH, Butler J, Kirk W. Mechanical effect of lung distention with positive pressure on cardiac function. Am Rev Respir Dis. 1981 Oct;124(4):382-6.
- 20. Berendes E, Lippert G, Loick HM, Brüssel T. Effects of positive end-expiratory pressure ventilation on splanchnic oxygenation in humans. J Cardiothorac Vasc Anesth. 1996 Aug 1;10(5):598-602.
- 21. Muench E, Bauhuf C, Roth H, Horn P, Phillips M, Marquetant N, Quintel M, Vajkoczy P. Effects of positive end-expiratory pressure on regional cerebral blood flow, intracranial pressure, and brain tissue oxygenation. Crit Care Med. 2005 Oct 1;33(10):2367-72.
- 22. Parker JC, Hernandez LA, Peevy KJ. Mechanisms of ventilator-induced lung injury. Crit Care Med. 1993 Jan;21(1):131-43.
- 23. Dreyfuss D, Saumon G. Ventilator-induced lung injury: lessons from experimental studies. Am J Respir Crit Care Med. 1998 Jan 1;157(1):294-323.
- 24. Eisner MD, Thompson BT, Schoenfeld D, Anzueto A, Matthay MA, Acute Respiratory Distress Syndrome Network. Airway pressures and early barotrauma in patients with acute lung injury and acute respiratory distress syndrome. Am J Respir Crit Care Med. 2002 Apr 1;165(7):978-82.
- 25. Daoud EG. Airway pressure release ventilation. Ann Thorac Med. 2007 Oct;2(4):176.
- 26. Daoud EG, Farag HL, Chatburn RL. Airway pressure release ventilation: what do we know? Respir Care. 2012 Feb 1;57(2):282-92.
- 27. Wrigge H, Zinserling J, Neumann P, Muders T, Magnusson A, Putensen C, Hedenstierna G. Spontaneous breathing with airway pressure release ventilation favors ventilation in dependent lung regions and counters cyclic alveolar collapse in oleic-acid-induced lung injury: a randomized controlled computed tomography trial. Crit Care. 2005 Dec;9(6):R780.
- Putensen C, Mutz NJ, Putensen-Himmer G, Zinserling J. Spontaneous breathing during ventilatory support improves ventilation– perfusion distributions in patients with acute respiratory distress syndrome. Am J Respir Crit Care Med. 1999 Apr 1;159(4):1241-8.
- 29. Varpula T, Valta P, Niemi R, Takkunen O, Hynynen



M, Pettilä V. Airway pressure release ventilation as a primary ventilatory mode in acute respiratory distress syndrome. Acta Anaesthesiol Scand. 2004 Jul;48(6):722-31.

 Esteban A, Frutos F, Tobin MJ, Alía I, Solsona JF, Valverdu V, Fernández R, de la Cal MA, Benito S, Tomás R, Carriedo D. A comparison of four methods of weaning patients from mechanical ventilation. N Engl J Med. 1995 Feb 9;332(6):345-50.

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